INTRODUCTION
Methanol—also referred to as methyl alcohol—is used widely in commerce and industry. Methanol toxicity is due to the formation of formaldehyde and formic acid. Methanol ingestion can also lead to acute pancreatitis and metabolic acidosis. Brain injury can also occur as ischemia or hemorrhage. The report is a 23-year-old male with methanol intoxication with complications of acute pancreatitis, alcoholic ketoacidosis, and intracranial hemorrhage.

Key words: methanol intoxication, acute pancreatitis, alcoholic ketoacidosis, intracranial hemorrhage

ABSTRACT
Background: Methanol intoxication occur after accidental or suicidal ingestion and the toxicity is due to the formation of formaldehyde and formic acid. Methanol ingestion can also lead to acute pancreatitis and metabolic acidosis. Brain injury can also occur as ischemia or hemorrhage. The report is a 23-year-old male with methanol intoxication with complications of acute pancreatitis, alcoholic ketoacidosis, and intracranial hemorrhage.

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INTRODUCTION
Methanol ingestion cause high anion gap metabolic acidosis from the production of formic and lactic acid and CNS disturbance ranging from inebriation and drowsiness to obtundation, seizure, and coma. Selective toxicity of the optic nerve and basal ganglia are well known features. Methanol ingestion can also lead to acute pancreatitis. Acute pancreatitis is a sudden inflammation of the pancreas characterized by a discrete episode of abdominal pain and elevated serum amylase and lipase levels. It can have severe complications and high mortality despite treatment, while mild cases are often successfully treated with conservative measures.

We report a patient with methanol intoxication presented with severe high-anion-gap acidosis metabolic and intracerebral bleeding.

CASE PRESENTATION
A 23-year-old male was admitted to the Emergency because of severe vomiting since 8 hours before admission accompanied with abdominal pain that spread to the back. He also suffered from shortness of breath since 2 hours before admission. He had suffered from headache 1 day before admission and started to feel nausea 12 hours before admission, followed by frequent vomiting containing residual food and fluid. (His mother said that he vomit more than 10 times). According to his neighbor, he had a party and drank alcohol 2 days before admission. Two of his friends have same symptoms and died in hospital. The patient had a habit to consume alcohol since 6 years ago.

On physical examination he looked severely ill with GCS 234, pupils were isocore with...
positive light reflex, blood pressure was 90/60, pulse rate 120 bpm, respiratory rate 40 x / min and axillar temperature 37.9 °C.

Laboratory results showed leukocyte 35,100 u/L, Potassium 8.24 mmol/L, Blood sugar 337 mg/dl, Amylase 187 U/l, Ureum 77 mg/dl, Creatin 2.31 mg/dl, and severe metabolic acidosis with pH 6.991, BE -24.9, anion gap 30.

The patient was transferred to ICU and rehydrated with NS 2l/hour and after the hypotension was successfully treated, insulin bolus 10u IV was given and continued with Actrapid™ 5U/hour. Emergency hemodialysis was planned but refused by his family. Natrium Bicarbonate 100 mg and Calcium gluconates 1000 mg was also slowly given intravenously to manage the metabolic acidosis and the hyperkalemia. Folic acid 1200 mcg, pantoprazole 30 mg and thiamine 3x200 mg was also administered.

The hemodynamic condition was improved markedly following these measures. The pH and kalium levels were back to normal. Despite this, the patient remained unresponsive and became unconscious. Because of the initial high level of methanol (124.3 mg/dl) and the severity of acidosis, severe neurological damage was to be expected.

CT scan was performed and showed 48 ml intracerebral hemorrhage in right fronto parietal lobe with perifocal edema.

Vitamin K (3x10 mg), mecobalamin (3x500mcg), and mannitol infusion 250 ml in 24 hours was given. After 8 days in ICU, the patient was moved to the Stroke Unit for CVA management, but his family decided to took him home because of financial problem. He died at home after 3 days.

DISCUSSION
Criteria for therapy initiation in patients with known or suspected methanol poisoning:
• Plasma methanol >20 mg/dL or
• Recent history of methanol ingestion in toxic amounts and an osmolar gap > 10 mOsm/L or
• Suspected methanol ingestion with at least 2 of the following: Arterial pH <7.3, Serum bicarbonate <20 mmol/L and Osmolar gap >10 mOsm/L.

The clinical features of methanol intoxication usually present after a latent period of 12-24 hours following methanol ingestion. This latent period corresponds to the time needed to convert methanol to formic acid by alcohol dehydrogenase in the liver, the metabolite responsible for the acidosis and the toxic effects. This patients had clinical symptoms 24 hours after drinking.

Alcoholic ketoacidosis causes nausea, vomiting, and abdominal pain. The patient came with hyperketonemia, high anion gap metabolic acidosis but without significant hyperglycemia.

Diagnosis is by history and findings of ketoacidosis without significant hyperglycemia. Alcoholic ketoacidosis is a metabolic complication of alcohol use and starvation characterized by hyperketonemia and high anion gap metabolic acidosis without significant hyperglycemia. Alcoholic ketoacidosis is attributed to the combined effects of alcohol and starvation on glucose metabolism.

Clinical features of acute pancreatitis (abdominal pain and vomiting) together with elevation of plasma concentrations of pancreatic enzymes (amylase, lipase) are the cornerstones of diagnosis. Pancreatic enzymes are released into the circulation during an acute attack; blood levels peak early, and decline over 3–4 days.

Alcohol use (approximately 35%) is a major cause of acute pancreatitis. Most commonly, the disease develops over 5-15 years; alcoholics are usually admitted with an acute exacerbation of chronic pancreatitis. Several case reports have described sole large alcohol load to precipitate a first attack. This features correspond to our patient; he had drunk alcohol since 6 years ago and came with abdominal pain and severe vomiting and elevated amylase.

Brain injury in methanol toxicity is characterized by lesions affecting both basal ganglia and subcortical regions. These lesion could be ischemic/necrosis or hemorrhage, selectively affecting bilateral putamen. It has been suggested that the putamen is at particular risk because of its high metabolic demand and located in end-zone of vascular perfusion. The precise mechanism of necrosis and hemorrhage in case of methanol toxicity remains a matter of debate. It may represent a direct toxic effect of methanol and its metabolite formic acid - as well as secondary injury to anoxia and acidosis. More recent study suggest that methanol induced cerebral vasospasm as a consequence of a large rise in intracellular calcium. These events could play a crucial role in methanol-induced cerebral edema, brain ischemia/necrosis and hemorrhage. It is also demonstrated that methanol elevates calcium ions in cerebral vascular muscle cells.
CONCLUSION

This case is a patient with methanol intoxication presented with severe high-anion-gap acidosis metabolic, complicated with intracerebral bleeding and acute pancreatitis. The treatment was supportive, acidosis metabolic correction, rapid glucose regulation with insulin. Patient was discharged because of financial problem and died at home.